68 The Motor System and Gait

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Definition

Loss of muscle strength may be complete *(paralysis, plegia)* or incomplete *(weakness, paresis)*. If one extremity is weak or paralyzed, it is termed a *monoparesis* or *monoplegia;* weakness or paralysis of one side of the body is a *hemiparesis* or *hemiplegia;* weakness or paralysis of both legs is a *paraparesis* or *paraplegia;* weakness or paralysis of all four extremities is a *quadriparesis or quadriplegia*; paralysis of like parts on the two sides of the body is a *diplegia.*

Muscle *atrophy* is a decrease in muscle volume or bulk. Conversely, muscle *hypertrophy* is an increase in muscle size. *Pseudohypertrophy* is an increase in muscle size due to infiltration by fibrous or fatty tissues and is usually associated with a decrease in strength.

Muscle *tone* refers to the tension present in relaxed muscle or to its resistance to passive movement. It may be decreased *(hypotonia)* or increased *(hypertonia)*. Within the latter group, *rigidity* is a state of steady, heightened, muscular tension equally present in opposing muscle groups. *Spasticity* is a velocity-dependent increase in tension of a muscle when it is passively lengthened.

Fasciculations are spontaneous visible muscle twitches due to contraction of the muscle fibers of a motor unit. *Fibrillations* are contractions of individual muscle fibers and are not visible through the skin. *Myokymia* is a condition characterized by continuous, slow, undulating contractions of muscle due to repetitive discharge of motor nerves.

Myotonia is a sustained and uncontrollable contraction associated with a delay in muscle relaxation.

Technique

Examination of the motor system has traditionally included assessment of muscle bulk, tone, power, and identification of any spontaneous activity. The tools required to accomplish this are little more than one's powers of observation, palpation, and judgment.

Observation

While the order in which the various aspects of the examination are covered can be individualized and tailored to the clinical situation, one convenient method is to begin with the process of *inspection*. There is no need to wait for the patient to enter the examining room; much can be learned from observing how the patient arises from a seat, walks into the room, and gets on and off the examining table.

Once in the examining room, have the patient disrobe (preserving, of course, necessary modesty). Although this may seem obvious, it is not unusual for telltale signs to be cloaked by clothes. The patient can then be scrutinized for evidence of wasting, weakness, and abnormal movements. When looking for focal wasting, compare the bulk of a muscle with its neighbor and counterpart on the opposite side. Keep in mind the patient's general build (slender individuals may seem diffusely "wasted"). Since asymmetric development is not uncommon, using minor differences in measured circumference of limbs to determine atrophy is notoriously unreliable.

In conjunction with wasting, muscle weakness may impart certain appearances. Facial muscle weakness may be signaled by an ironing out of forehead wrinkles and/or flattening of the nasolabial fold, sagging of the lower eyelid, hollowing of the temples, or inability to close the eye fully. Sometimes mild weakness only becomes evident when the patient smiles; the angle of the mouth, instead of turning up, remains horizontal or depressed, producing a "snarl." Involvement of the eyelid elevators results in drooping of the lid (ptosis). A compensatory raising of the eyebrows may maintain the weak lid at a normal level, but is revealed by an increased number of forehead creases on the abnormal side.

As the muscles that act to brace the scapula to the thorax weaken, several characteristic changes occur. The hands, which in normal standing are held with the thumbs facing forward, tend to turn inward, causing the back of the hand to face front. Early on, one might detect a hump in the midportion of the trapezius as the muscle works to aid in scapular fixation. Viewed from the back, the lower medial border of the scapula may protrude out, or "wing." Since this finding is not always readily apparent in afflicted patients and yet can be mimicked by a comparable profile in normal thin individuals, it is helpful to have the patient lift the arms in front of the body, then lower them slowly. This maneuver will cause the scapular edge to "pop" out, accentuating any true winging. As shoulder–girdle strength deteriorates, the scapula actually slides laterally and upward, as if it were folding over the clavicle. With associated pectoral muscle wasting, the axillary fold, which normally runs straight up and down, becomes slanted diagonally inward.

The limbs can be similarly surveyed for loss of muscle bulk or abnormal posture. The wasted muscles have a flattened or scalloped contour, and when tensed, they do not develop the normal bulge. With weakness of thenar muscles in the hand, the thumb, instead of being held at right angles to the fingers, comes to lie in the same plane as them, producing a "simian" hand. If the other intrinsic hand muscles are affected, the hand assumes a clawlike deformity, the fingers being extended at the metacarpophalangeal joints and flexed at the interphalangeal joints. Muscular weakness in the lower extremity is often betrayed by alterations in gait, described below.

As one is going through the visual inspection, be alert for any spontaneous muscle activity, such as fasciculations or myokymia. Fasciculations may be difficult to see in obese patients or infants. It is said that they can best be appreciated with oblique lighting; sometimes lightly tapping the muscle

will activate them. Searching for fasciculations in the tongue may be significant diagnostically but is prone to misinterpretation. When protruded, the tongue might appear to be quivering due to contraction of the small intrinsic muscles. Therefore, one should have the patient partly open the mouth and view the tongue as it rests quietly on the floor of the mouth. Myokymia, unlike the brief twitch of fasciculation, is of a slower and more prolonged nature, so the muscle surface seems to undulate.

Palpation

Direct *palpation and percussion* can at times be a useful adjunct in appraising the muscle. With degeneration and atrophy, muscle loses its normal texture and becomes soft and flabby. Enlarged muscles due to true hypertrophy are firm and resilient, whereas in pseudohypertrophy, such as occurs in some forms of muscular dystrophy, the muscle takes on a rubbery or "woody" consistency. In patients who complain of muscle tenderness and nodules, palpation can help ascertain that these findings arise from the muscle itself and not the overlying tissues.

Percussion of muscle usually has a more limited role in the examination, yet also may produce diagnostic responses. One such response is myotonia. This phenomenon is readily demonstrated in the thenar eminence, where a sharp tap with a percussion hammer causes adduction of the thumb that persists for several seconds before relaxation occurs. Cooling the extremity may be necessary to bring out the myotonia. Other muscles, such as the deltoid or tongue, can be percussed, and if involved, will show a depression that slowly disappears. Functionally, myotonia can be uncovered by simply shaking hands and noting the patient's inability to release quickly. Remember that normal muscle will also react to being struck, but the contraction is brief and relaxes immediately. Furthermore, myotonia should not be mistaken for myoedema, a reaction seen in hypothyroidism and other metabolic disturbances. Here, percussion causes a dimpling in the muscle that radiates outward, followed by development of a small mound at the point of impact that remains for a few seconds.

Judgment

While some clues to muscle tone can be gleaned from inspection and palpation, assessment hinges on *judging* the resistance of muscle to passive movements. Implicit in this criterion is that the patient be relaxed. Nevertheless, demanding that the patient "relax" often produces, not unexpectedly, the opposite result. Instead, by making the testing an inobtrusive part of the exam, a sufficient degree of relaxation can usually be attained. The patient may need to be gently instructed to go "loose" or "floppy" or make the limb "like spaghetti." Tone can then be tested by moving a part through its full range of motion, initially slowly and then at varying speeds. The upper extremities are commonly checked by flexing and extending the elbow, rotating the hand at the wrist, grasping the wrist, and shaking the hands or grasping the hands and shaking the arms. In the lower limbs, one can pump the knee or, with the patient supine and the examiner's hand under the patient's knee, quickly lift the knee off the table, noting if the heel slides up the table (normal) or actually rises off (hypertonia).

A hypotonic limb feels limp and hyperextensible, as if one were shaking a wet noodle. With spasticity, increasing tension develops during rapid movement that may suddenly melt away (the "clasp-knife" effect). Since opposing muscles are involved to differing degrees, the resistance is usually greater in one direction than the other. Moreover, spasticity is a velocity-dependent phenomenon, so resistance at slow speeds will be even. Rigidity, another state of increased tone, differs from spasticity in that opposing muscles are affected equally, providing a constant "pull" through the entire range of motion and at any speed. In certain extrapyramidal disorders (e.g., Parkinson's disease), one may also detect a superimposed rachety sensation on passive motion, termed *cogwheeling*. Often this type of rigidity is most easily perceived at the wrist and can be intensified by repetitive movements of the opposite side (e.g., tapping the hand on the knee, opening and closing the fist). A commonly confusing clinical finding seen in patients with diffuse cerebral dysfunction is *gegenhalten* or *paratonia*. Here the patient seems actively to oppose any passive motion, despite the examiner's exhortations to relax.

Evaluation of Muscle Strength

Any suspicions of weakness that arise from other sections of the examination can be resolved by *evaluation of muscle strength*. This fundamental aspect of motor testing is worth mastering, for it allows one to determine both the degree and distribution of weakness present.

Muscle power can be judged either by manual testing of individual muscles or by having the patient perform maneuvers that stress certain muscle groups. These two methods are not mutually exclusive and should be used in a complementary fashion.

Individual muscles can be tested by instructing the patient either to resist the efforts of the examiner to move a fixed part or to initiate movement against the examiner's resistance. While the latter technique may be best for studying very weak muscles, the former is generally simpler for the patient to understand and cooperate with, and enables the physician to better isolate the muscle being tested.

In actuality, isolating the action of a single muscle is extremely difficult, since other functionally alike muscles contribute to every movement. Nevertheless, by proper positioning, the prevailing activity of a muscle can usually be discerned. For example, hip extension with the knee extended involves participation of the gluteus maximus and hamstrings; if the knee is flexed, only the gluteus maximus acts to extend the hip. Moreover, attention to positioning is important in preventing the employment of an accessory muscle to substitute for a weakened one (e.g., with biceps weakness, the patient may partially pronate the forearm and utilize the brachioradialis). In contrast, a normal muscle may appear weak if it is placed at a mechanical disadvantage (e.g., in the presence of a wrist drop, the finger flexors may seem weak unless the wrist is extended). This pitfall can be partially avoided by fixating the proximal side of the joint when the distal parts are being tested. Finally, it is good to examine very weak muscles in an altitude where the effects of gravity are eliminated.

Hand in hand with a system of examination must be a means of recording and communicating the results. While muscle power can be graded in various ways, perhaps the most widely accepted is the Medical Research Council (1976) classification, which rates strength on a scale of 0 to 5 as

follows: 0, no movement; 1, trace of contraction; 2, active movement when gravity is eliminated; 3, active movement against gravity; 4, active movement against gravity and resistance; 5, normal strength.

Routine testing of every muscle is clearly not practical. Probably a more reasonable screen would be to examine strength at each major joint through its principal movements, focusing the investigation as clinically indicated. The reader is referred to DeJong (1979), Mayo Clinic (1981), and Medical Research Council (1976), several excellent texts with "how to" descriptions of individual muscle evaluation.

Although manual testing is essential when trying to determine the exact distribution of impairment, it has the disadvantage of being somewhat time-consuming and insensitive to mild grades of weakness, particularly in the powerful muscles of the lower extremities. *Functional testing*, which admittedly looks only at groups of muscles, is often better at uncovering slight paresis and monitoring the progression of a disease. As an example, very mild upper-limb weakness may be disclosed by having the patient hold the arms extended out front, palms up, eyes closed, for about a minute. If strength is reduced, the arm will pronate and begin to drift down.

Pelvic girdle musculature can be assessed by asking the patient to carry out several tasks including arising from a chair and getting up from a squatting position. With hip weakness there may be hesitation during the movement, or patients may need to use their arms or some momentum to boost themselves. Another test of pelvic girdle strength, one that is especially applicable to children, is arising from a sitting position on the floor. Classically, weak patients turn on their side, pull the knees up under the body, and rest on hands and knees. They then straighten the knees and lift the buttocks in the air while remaining flexed at the hips. Finally, they straighten at the hips, occasionally placing a hand on the thigh for an extra push. This is known as Gower's maneuver. Distal weakness in the legs may manifest itself by difficulty walking on one's heels when the ankle dorsiflexors are involved or walking or hopping on the toes if ankle plantar flexors are weak.

Indeed, the manner in which one walks attests to the functioning of multiple levels of the neuraxis. As mentioned, the clinician should get into the habit of watching the patient walk into and around the examining room. Look at the whole person—that is, not only the legs but also the *posture* (stooped? lordotic?), the *arm swing* (free or reduced? symmetric or not?), and any *associated movements* (e.g., chorea or dystonia). Note the length and speed of individual steps. Observe if the patient assumes a broad base or keeps eyes glued to the ground. Listen to the gait—for the flopping sound of a foot drop, the scraping sound of a hemiparetic gait, or the stamping sound of an ataxic gait. Inspect worn places on the patient's shoes. During more formal testing, ask the patient to turn quickly on command (counting the number of steps required to turn); to walk as if on a tightrope, one foot directly in front of the other (a good way to detect ataxia); and/or to walk on the heels or toes.

One must be aware of factors such as contractures, involuntary movements, changes in tone, proprioceptive deficits, pain, and psychiatric disorders that can interfere with patient performance despite normal strength. Certainly, care should be taken in interpreting any apparent weakness if pain is present. Unlike the diminished but constant resistance offered by the paretic individual, the patient with pain often displays poorly sustained efforts that abruptly give way. A similar finding can be noted in patients with hysterical motor weakness. Hysterical weakness may be suspected by feeling the contraction of antagonist muscles while testing the agonist. Or the patient may be observed normally doing an activity that requires the action of an allegedly enfeebled muscle (e.g., easily stepping onto the examining table yet unable to lift the thigh on request).

Basic Science

Even the most effortless movement involves a highly complex and incompletely understood integration of multiple neural circuits. What follows is merely a simplified, schematic review of the motor system.

There is evidence to suggest that the idea to perform a movement originates in the motor association areas of the frontal lobe. The subsequent planning and initiation of the action, though, require input from the basal ganglia and cerebellum. Both structures receive information from the frontal association areas and funnel their output to the motor cortex via the thalamus. Moreover, they are constantly kept abreast of ongoing motor activity not only through ascending sensory pathways (either direct or indirect) but also by collaterals of corticospinal tract fibers. These networks allow the basal ganglia and particularly the cerebellum to coordinate muscle activity so that movements are smooth and accurate. Not surprisingly, when the cerebellum and basal ganglia are damaged, movements become clumsy, the spatial and temporal patterning of muscle contraction is disrupted, and involuntary movements appear.

The motor cortex is thought responsible for sending the final commands to execute a movement. The message passes downstream via several descending pathways, perhaps the best known being the corticospinal or pyramidal tract. The constituent fibers of this system arise in approximately equal numbers from the motor cortex (area 4), the adjacent frontal area 6, and sensory areas in the parietal lobe. These fibers descend nonstop through the posterior limb of the internal capsule, basis pedunculi, and medullary pyramids, with the majority decussating in the caudal portion of the medulla to form the lateral corticospinal tract of the cord. They are destined to end directly on either motor neurons, interneurons, or sensory neurons.

Not to be ignored, however, are the "extrapyramidal" descending pathways. Indeed, experimental lesions of the pyramidal tract at the medullary pyramids causes hypotonia and derangement only of fine distal movements. These other tracts also contain cortical fibers, but make connections through such subcortical way stations as the basal ganglia, cerebellum, and brainstem nuclei. Except for the cerebellum, most of these components are inhibitory in total effect and, if injured, usually result in spasticity or rigidity. In actuality, naturally occurring lesions tend to affect a combination of descending fibers; the clinical features that result are generally said to reflect "upper motor neuron" damage.

The final common pathway through which the central nervous system (CNS) controls skeletal muscle action are the motor neurons of the spinal cord and cranial motor nuclei. Each motor neuron innervates a group of muscle fibers and, with the fibers, constitutes a motor unit. Discharge of the motor neuron results in contraction of its muscle fibers.

Whether or not these lower motor neurons fire is contingent on the summation of descending and local reflex information that impinge on them. One basic circuit that

participates in local reflex activity and contributes significantly to muscle tone is the gamma loop. Skeletal muscle contains specialized receptors called *spindles* that signal the spinal cord when the muscle is stretched. Within each spindle is a small muscle fiber innervated by a gamma, or small-diameter motor fiber, which sets the spindle's tension. The higher the tension, the more sensitive the spindle is to stretch further, and vice versa. Thus, the gamma system can bias the reactivity of the spindle and regulate the local afferent influence on the motor neuron. Moreover, the gamma system itself is modulated by descending impulses, both tonic and phasic in nature. Many disorders of CNS motor function (e.g., spasticity, rigidity) can be understood in terms of imbalance in this system.

Finally, all voluntary movement is guided by continuous sensory feedback to the spinal cord and higher centers. Since the sensory and motor systems are inextricably tied together, impairment of the former may disturb many aspects of normal movement.

Clinical Significance

Realizing that the components of the motor system work as a unit, it would be an oversimplification strictly to attribute certain signs to specific anatomic systems. Nevertheless, characteristic changes in muscle tone, strength, and gait can provide direction toward the location and nature of a pathologic process (see Table 68.1).

Features of *upper motor neuron* (corticospinal) *system dysfunction* can occur with lesions of the motor cortex or descending pathways in the corona radiata, internal capsule, brainstem, or spinal cord. Motor findings will be contralateral to the neural damage if above the decussation of the pyramids, ipsilateral if below. Depending on the site and extent of the lesion, weakness can vary anywhere from a slight pronator drift and clumsiness with skilled distal movements to complete plegia. Entire extremities or discrete movements, rather than particular muscles, are involved, with relative sparing of flexors and internal rotators in the upper extremities and extensors and external rotators in the lower extremities. These same muscles tend eventually (though not inevitably) to develop an increase in tone, often with spasticity. Atrophy is not prominent early, although some wasting may occur over time owing to disuse. Gait, however, is frequently altered due to the increased tone and extensor predominance in the leg. With hemiparesis, the arm tends to be held tightly to the side, flexed and stiff without the normal swing. The leg is thrown outward from the hip and circumducted. In doing so, the toe of the shoe is dragged and may be noticeably worn on the outer aspect. When both legs are involved, the gait may become scissor-like, each knee crossing in front of the other with every step.

Disease of the lower motor neuron engenders an entirely different clinical constellation. Weakness may be restricted to the distribution of a certain spinal segment or peripheral nerve or be more diffuse, as in a polyneuropathy that tends to have its maximal effect on distal limb muscles. Muscle atrophy is often pronounced, sometimes associated with hypotonia. With motor neuron and root irritation, spontaneous muscle fasciculations may be seen. If foot drop due to weakness of ankle dorsiflexors is present, the gait may take on a "steppage" quality. In order to avoid dragging the toe, the patient compensates by lifting the leg high, with the toe hitting the ground first. This may resemble a gait seen with profound sensory loss where the patient stamps the foot down to gain more sensory input (in this instance, though, the entire sole tends to strike at once).

Primary muscle disease may find expression in several patterns of weakness, none of which correspond to either a specific spinal segment or a nerve. Commonly, the involvement is symmetric and proximal, although other selective forms occur (e.g., facioscapulohumeral syndromes). There may be tenderness, wasting, or even pseudohypertrophy. Tone, in the absence of contractures, can be normal or decreased. In patients with pelvic girdle weakness, the gait may assume a waddling nature because of the inability to fix the pelvis. The patient walks with a marked lordosis, throwing the hips from side to side to shift the weight of the body.

Injury to the cerebellum or basal ganglia, while not measurably reducing muscle power, can definitely impair motor function. Cerebellar "signs" can result from disease of the cerebellum itself or its inflow or outflow tracks in the brainstem, and are ipsilateral to the lesion. While hypotonia and an ill-defined muscle fatigability may occur, the principal manifestations of *cerebellar dysfunction* are disturbances of coordination and gait. Particularly with midline cerebellar lesions, the gait becomes broad based, unsteady, staggering, and lurching. The patient is unable to tandem walk and may persistently sway to one side if damage extends into a cerebellar hemisphere. The ataxic gait of cerebellar disease should not be confused with the bizarre ambulatory exhibitions of psychogenic origin. These "hysterical" gaits are recognizable by their inconsistency, varying from moment to moment and often requiring more coordination than normal.

Basal ganglia disorders can also profoundly interfere with normal ease of movements. Certain conditions present in a "parkinsonian" manner characterized by an increase in tone with rigidity and cogwheeling, a paucity of and slowness in initiating movements, and reduction or loss of many normal associated movements—all in the absence of decreased muscle power or atrophy. The gait reflects these deficits. The patient walks with a stooped posture, hips and knees slightly flexed, with little or no arm swing. Although the beginning steps may be quite slow, there is a tendency for the pace to

Table 68.1
Characteristic Clinical Motor Syndromes

	Lower motor neuron	Corticospinal	Extrapyramidal	Cerebellar
Atrophy	Present	Absent	Absent	Absent
Tone	Decreased	Increased (spasticity)	Increased (rigidity)	May be decreased
Weakness	Present (individual muscles affected)	Present (groups of muscles affected)	Absent	Absent
Fasciculations	Present	Absent	Absent	Absent
Gait	Steppage, waddling	Spastic (circumduction or scissoring)	Festination, shuffling	Broad based, ataxia

quicken as the patient shuffles forward (festination). The patient may turn en bloc, doing so in several small steps; sudden changes of direction cannot be done easily. A contrasting picture is seen with chorea. The patient may have a decrease in tone with marked hyperextensibility of the joints. The gait may be wide based and lurching, but more often is remarkable only for the excessive abnormal movements and posturing.

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